Papers

Analysis of the sociodemography of gonorrhoea in Leeds, 1989-93

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Abstract

Objective: To investigate the epidemiology of gonorrhoea in an urban area in the United Kingdom.
Design: Analysis of all cases of gonorrhoea with regard to age, sex, ethnic group, and socioeconomic group with 1991 census data as a denominator.
Setting: Leeds, a comparatively large urban area (population around 700 000) in the United Kingdom.
Subjects: All residents of Leeds with culture proved cases of gonorrhoea during 1989-93.
Main outcome measure: Relative risk of gonorrhoea.
Results: Sex, age, race, and socioeconomic group and area of residence were all independently predictive of risk of infection. Young black men aged 20-29 were at highest risk, with incidences of 3-4% per year. Black subjects were 10 times more likely than white subjects to acquire infection, and subjects from the most deprived socioeconomic areas were more than four times more likely than those from the most affluent areas to acquire infection.
Conclusions: Different ethnic and socioeconomic groups vary in their risk of infection with gonorrhoea within an urban area. Targeted interventions and screening to reduce the incidence of sexually transmitted disease are now priorities.

Introduction

Sexually transmitted diseases remain a major public health problem worldwide. In England sexual health was one of the key areas in the Health of the Nation strategy,[1] and a target was set of reducing the incidence of gonorrhoea in 1990 of 61 new cases per 100 000 population by 20% in 1995.

Gonorrhoea was the first sexually transmitted disease in which the dynamics of transmission were studied in depth using mathematical models.[2 3] These studies suggest the existence of core groups in maintaining the endemicity of gonorrhoea; these groups have higher incidences of infection and levels of sexual activity than the general population. The first empirical confirmation of such core group transmission was obtained by analysing the area of residence of people with gonorrhoea in upstate New York, and this showed an intense concentration in the inner city, relative risks for the central core area being 19.8 for men and 15.9 for women.[4] Further data from the United States have shown young black men and women to be at increased risk of infection.[5 6] In 1991 data on teenagers aged 15 to 19 in different regions of the United States showed that black men had a 73.3-fold increased risk compared with white men and black women a 23.3-fold increased risk compared with white women.[6] To our knowledge, no analysis has determined whether these differences in risks are attributable to socioeconomic factors rather than ethnic group or a combination of both.

The 1991 census in the United Kingdom provided detailed information of the population and for the first time included data on ethnic group.[7] We therefore studied the epidemiology of gonorrhoea within a defined large urban area, focusing on age, ethnic group, and socioeconomic variables.

Subjects and methods

We recorded details of all people resident within the boundaries of Leeds Health Authority who presented with culture confirmed gonorrhoea from 1 April 1989 to 30 September 1993 at this hospital, which is the only sexually transmitted disease clinic serving the Leeds city population of around 700 000 people. Patient information recorded included sex, age, date of birth, whether the gonorrhoea had been acquired heterosexually or homosexually, clinic number (used to identify people with recurrent episodes), and ethnic group. During the study ethnic group was assigned by reception staff using the Office of Population Censuses and Surveys' classification (this was replaced in 1995 by self assigned ethnic group). For the study analyses this classification was simplified to the categories white, black (includes black Caribbean, black African, black other), Asian (includes Indian, Pakistani, Bangladeshi, Chinese, Asian other), or other. All other microbiology laboratories and sexually transmitted disease clinics within a 20 km radius agreed to provide the same data for culture confirmed cases of gonorrhoea in people who were resident within the Leeds city boundaries and presented to their services.

We used the Super Profile classification as an indicator of socioeconomic status[8] as this had been used to analyse morbidity and mortality in the population of Yorkshire.[9] The Super Profile classification we used is a 10 cluster group classification of enumeration district areas of similar socioeconomic status on the basis of 120 census variables which are transformed using principal component analysis. The 10 socioeconomic categories are subsequently referred to as groups 1 to

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10. Deprivation indices such as the Jarman, Townsend, and Carstairs indices are strongly correlated with Super Profiles ($r=0.822$, 0.867, 0.911 respectively).[8] Postcodes were used to derive the socioeconomic group of patients on the basis of the enumeration district in which they lived. Denominators to calculate the incidence were taken from the 1991 census.

Analyses

Crude incidences and their exact asymmetric 95% confidence intervals were obtained by sex, age, race, and socioeconomic group using a method described by Miettinen.[10]

Logistic regression was used to model the incidence of infection using sex, age, race, and socioeconomic group as independent variables.[11 12] Relative risks with 95% confidence intervals were used to measure the likelihood of infection associated with values of the independent variables, with white men aged 15-19 living in enumeration districts classed as socioeconomic group 1 as the reference group.

Results

Time series and number of episodes

Between 1 April 1989 and 30 September 1993, 1416 people presented with a total of 1664 gonococcal infections. Ninety seven per cent of cases were treated at the Leeds General Infirmary. Only 105 cases (6.3%) occurred in homosexual men, and these are included and not distinguished further in the analyses. During the study the incidence of gonorrhoea fell by 50%. This was found equally in men and women and in white and black subjects. The peak incidence of cases was always seen in the third quarter (July to September) of each year. The proportion of subjects with repeat infections was 29% (85/294) for black men, 19% (17/89) for black women, 10% (52/521) for white women, and 7% (32/491) for white men.

Crude incidences

The crude incidences of gonococcal infection by sex, age, ethnic group, and socioeconomic group are shown in table 1. Those under 15 years old, those over 55 years old, and those in socioeconomic group 2 are excluded from table 1 owing to small numbers. Men had a higher rate of infection than women—54.5 per 100 000 (95% confidence interval 50.8 to 58.4) compared with 38.5 per 100 000 (35.5 to 41.7) respectively. Peak incidences across the age distribution occurred in men aged 20-24 years (268.4 per 100 000) and women aged 15-19 (250.3 per 100 000). The most striking result from this preliminary analysis is that the overall incidence in black subjects was 793.4 per 100 000 (716.3 to 876.3). This is 22 times greater than the rate in white subjects and 62 times greater than that in Asians. All of these differences were significant ($P<0.05$).

Rates of infection varied significantly between socioeconomic groups. People living in areas that fell into the five most prosperous categories of this classification had similar and comparatively low rates of infection, ranging from 23.0 to 32.3 per 100 000 in men and from 17.3 to 21.1 per 100 000 in women. These more affluent areas generally lie outside Leeds city centre. The incidence of infection among people living in areas that were in the five most deprived groups of the socioeconomic classification had comparatively high rates of infection that varied considerably. Those living in areas classed as group 8 and group 10 had the highest rates of infection: 293.0 and 178.8 per 100 000 in men and 208.3 and 125.3 per 100 000 in women respectively. Typically, these neighbourhoods were inner city areas with high proportions of ethnic minority groups; nearly half of all Leeds's black and Asian populations live in group 8 enumeration districts.

Table 1 Incidence of gonorrhoea by sex, age, ethnic group, and socioeconomic group in Leeds, 1989-93

Category	Incidence per 100 000 (95% CI)	No of people infected
Men		
Age (years):		
15-19	128.5 (107.1 to 153.0)	126
20-24	268.4 (239.4 to 299.9)	311
25-29	170.8 (148.1 to 196.0)	202
30-34	75.2 (59.8 to 93.5)	81
35-44	29.7 (22.9 to 38.3)	60
45-54	11.5 (6.9 to 18.0)	19
Ethnic group:		
White	54.4 (49.7 to 59.4)	491
Black	1887.9 (1679.9 to 2114.0)	294
Asian	38.3 (20.4 to 65.5)	13
Other	99.3 (32.2 to 231.6)	5
Socioeconomic group:		
1	23.0 (14.2 to 35.2)	21
3	25.3 (15.6 to 38.6)	21
4	27.0 (19.2 to 36.9)	39
5	32.3 (24.3 to 42.1)	54
6	43.7 (34.0 to 55.3)	69
7	87.8 (63.3 to 118.6)	42
8	293.0 (257.6 to 331.8)	248
9	81.4 (61.6 to 105.4)	57
10	178.8 (154.8 to 205.4)	199
Women		
Age (years):		
15-19	250.3 (219.4 to 284.3)	236
20-24	174.6 (151.9 to 199.6)	213
25-29	72.1 (57.9 to 88.7)	89
30-34	40.1 (29.1 to 53.8)	44
35-44	9.4 (5.7 to 14.7)	19
45-54	1.8 (0.4 to 5.2)	3
Ethnic group:		
White	56.2 (51.5 to 61.3)	521
Black	558.0 (448.4 to 686.3)	89
Asian	3.0 (0.1 to 16.4)	1
Other	0 (0 to 82.5)	0
Socioeconomic group:		
1	17.3 (9.9 to 28.1)	16
2	18.7 (10.7 to 30.4)	16
4	21.1 (14.4 to 30.0)	31
5	20.7 (14.4 to 28.8)	35
6	44.3 (34.7 to 55.7)	73
7	71.1 (49.5 to 98.8)	35
8	208.3 (178.0 to 242.3)	168
9	62.0 (45.4 to 82.7)	46
10	125.3 (105.9 to 147.2)	148

Logistic regression

As the univariate analyses had suggested that sex, age, ethnic group, and socioeconomic group were all related to incidence these variables were used as predictors of incidence in a regression model. The best fitting model contained eight significant effects: all four variables of the first model plus the interactions of sex by age, age by

socioeconomic status, age by ethnic group, and sex by ethnic group. The results of this model are shown in figure 1, which shows the relative risks of gonococcal infection by sex, age, and ethnic group for socioeconomic groups 1, 6, and 8. The sex by age by race patterns of risk that emerge for these three socioeconomic groups are similar, although the relative risks are different, with a greater than fourfold increased risk between the most affluent and most deprived socioeconomic groups. Indeed, results from a model not fully presented here suggest that, on average, after controlling for sex, age, and socioeconomic group, black subjects in Leeds were more than 10 times likely than white subjects and about 50 times more likely than Asian subjects to have had one or more episodes of gonorrhoea during the study.

In most age groups in all socioeconomic groups the risk of infection for Asian women was significantly lower than that for white women. Similarly, although not significantly, Asian men generally had lower rates of infection than white men. On average, after controlling for sex, age, and socioeconomic group, white subjects in Leeds were nearly five times more likely than Asians to have had one or more episodes of gonorrhoea during the four and a half years of the study.

Discussion

Risk factors for gonorrhoea

We found large variations in the incidence of gonorrhoea among different groups of subjects within a large city in the United Kingdom. We believe that the discrete urban area and the open access services to sexually transmitted disease clinics in the United Kingdom will have made our degree of case ascertainment high. Previous data from the United Kingdom have shown variations in the incidence of gonorrhoea with age, sex, and geography.[13-15]

We found extremely high rates of gonorrhoea among young black subjects, with black men aged 20-29 at the highest risk. Underenumeration in the 1991 census preferentially occurred among those aged 20-29 and also to a degree among black compared with white subjects. However, this is likely to reduce the relative risk for black compared with white people aged 20-29 by a factor of only 0.97-0.95.[16] Observer assigned ethnic group may have misclassified people of mixed or other racial groups as black. Nevertheless, the incidences we found for black and white subjects and their respective differences are similar to published data from the United States.[5 6]

We looked for any independent contribution of socioeconomic group to risk of gonorrhoea—that is, independent of race and other factors. We used a validated method based on socioeconomic variables associated with small defined areas of residence. Although we clearly found that socioeconomic group as thus defined was an independent risk factor, this variable could represent an effect of social characteristics and networks within certain neighbourhoods.[17 18] The free confidential services of sexually transmitted disease clinics in the United Kingdom mean that it is unlikely to be related to the availability of treatment. Figure 1 shows that differences in risk by ethnic group persist across all socioeconomic or residential groups and that the size of increased risk between least and most afflu-

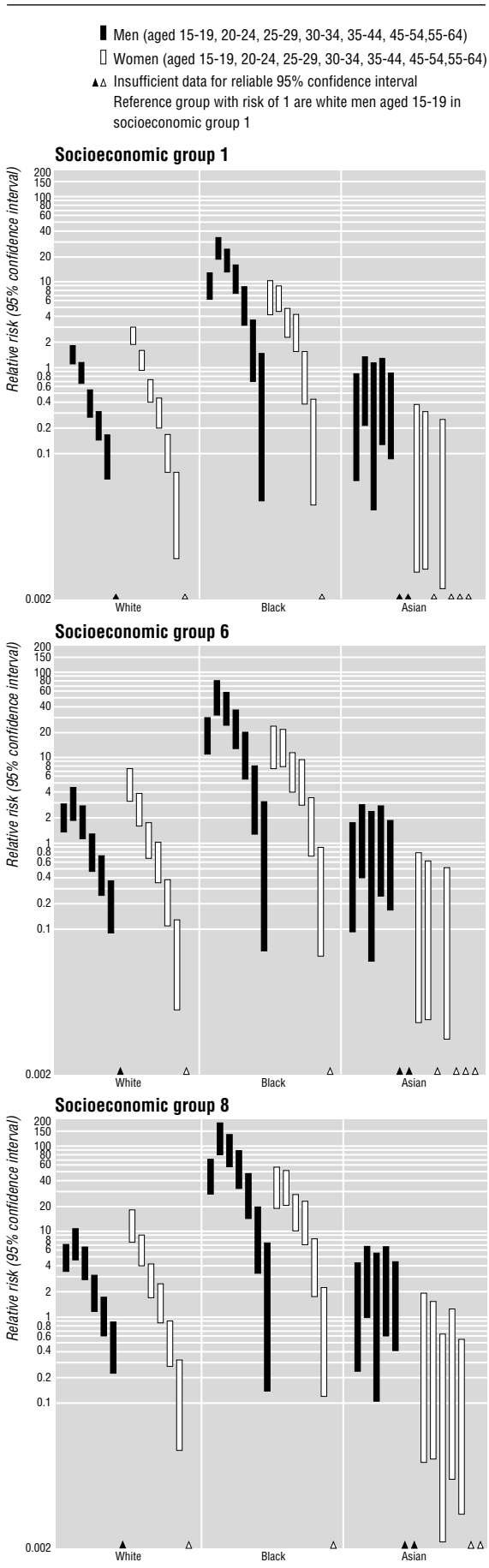

Fig 1 Relative risks of gonococcal infection (with 95% confidence intervals) by sex, age, and ethnic group in socioeconomic groups 1,6, and 8 (ln scale)

Key messages

- Ethnic group and socioeconomic group or area of residence are independent risk factors for gonorrhoea
- Ethnic group and factors associated with neighbourhood of residence may modulate sexual risk factors through cultural and behavioural mechanisms
- Sexual risk reduction and disease screening interventions targeted at groups at greater risk should now be evaluated

ent is far smaller than for that of people from different ethnic groups. The final geographical outcome of these contributor risk factors (mapping studies not shown) show a more complex pattern than reported from the United States.[4 5 17] Our use of small geographical units defined various areas with high and medium incidence of gonorrhoea across the city.

Cultural and behavioural mechanisms

Our analyses have allowed us to conclude that socioeconomic status is not the primary cause of variations in incidence associated with ethnic group. The national survey of sexual lifestyles in the United Kingdom showed that black men were significantly more likely than white or Asian men to report having their first sexual intercourse before the age of 16, but there was no significant difference between white, black, and Asian women.[19] Univariate analysis in the same survey suggested that black men reported having a greater number of sexual partners than did white men (median 8 *v* 4 respectively), but the number of observations was small and multivariate analysis was not performed (A M Johnson, personal communication). Such differences were not observed between white and black women. Ethnic influences probably affect sexual behaviour through cultural or contextual mechanisms,[20] although there is a dearth of research on this subject. However, we suggest that the beliefs and sexual behaviour of young black men as a group mediate high levels of risk activity and gonococcal acquisition and transmission within defined communities.

Future research should focus on effective interventions to reduce risk behaviour and gonococcal transmission. Knowledge of the geographical distribution of infection within urban areas can enable targeted programmes and screening to be developed. Culturally appropriate interventions that decrease sexual activity risk behaviours in inner city populations have been described.[21 22] Implementation of such programmes with evaluation of their medium term effects should now become a research priority.

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A salutary lesson

Family secrets

I once anaesthetised a patient whose husband had been unaware that she wore dentures until I asked her about them while he was sitting at her bedside. From that day to this I have never interviewed an adult patient without first asking any visitors to leave. In the age of open visiting hours it is increasingly common to have to do so. Some relatives, spouses, and mothers in particular, can be quite offended by this—"we have no secrets"—but as I discovered that day, people sometimes do, and my responsibility is to get the information I need from the patient.

Our modern caring and sharing society means that increasingly discussions and treatments are carried out with relatives present (even cardiopulmonary resuscitation, apparently, for goodness sake) on the assumption that nobody could possibly object to the involvement of a loving family member.

The moral of this story is that we should never make assumptions about other families' degree of intimacy on the basis of our own values. This seems to be growing in importance as partners and other family members encroach more and more on the doctor-patient relationship. On the whole, support during illness is a good thing and should be encouraged, but do we sometimes forget the patient in our desire to involve the family?

Hilary Aitken, *consultant anaesthetist, Redditch*

*We think patients should decide themselves who should be present at the interview, and mostly they will want their close friends or relatives there. Dr Aitken points out a possible difficulty in asking patients what they want. Any views?
Roger Robinson, *BMJ*

Gonorrhoea in inner London: results of a cross sectional study

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Abstract

Objectives: To estimate population based incidence rates of gonorrhoea in an inner London area and examine relations with age, ethnic group, and socioeconomic deprivation.
Design: Cross sectional study.
Setting: 11 departments of genitourinary medicine in south and central London.
Subjects: 1978 first episodes of gonorrhoea diagnosed in 1994 and 1995 in residents of 73 electoral wards in the boroughs of Lambeth, Southwark, and Lewisham who attended any of the departments of genitourinary medicine.
Main outcome measures: Yearly age, sex, and ethnic group specific rates of gonorrhoea per 100 000 population aged 15-59 years; rate ratios for the effects of age and ethnic group on gonorrhoea rates in women and men before and after adjustment for confounding factors.
Results: Overall incidence rates of gonorrhoea in residents of Lambeth, Southwark, and Lewisham were 138.3 cases yearly per 100 000 women and 291.9 cases yearly per 100 000 men aged 15-59 years. At all ages gonorrhoea rates were higher in non-white minority ethnic groups. Rate ratios for the effect of age adjusted for ethnic group and underprivilege were 15.2 (95% confidence interval 11.6 to 19.7) for women and 2.0 (1.7 to 2.5) for men aged 15-19 years compared with those over 30. After deprivation score and age were taken into account, women from black minority groups were 10.5 (8.6 to 12.8) times as likely and men 11.0 (9.7 to 12.6) times as likely as white people to experience gonorrhoea.
Conclusions: Gonorrhoea rates in Lambeth, Southwark, and Lewisham in 1994-5 were six to seven times higher than for England and Wales one year earlier. The presentation of national trends thus hides the disproportionate contribution of ongoing endemic transmission in the study area. Teenage women and young adult men, particularly those from black minority ethnic groups, are the most heavily affected, even when socioeconomic underprivilege is taken into account. There is urgent need for resources for culturally appropriate research and effective intervention to prevent gonococcal infections and their long term sequelae in this population.

Introduction

The sexual health of the nation is a key topic prioritised for improvement by the British government.[1] The incidence of gonorrhoea was chosen as a proxy indicator for trends in condom use and new cases of HIV infection.[2] Gonorrhoea merits prevention efforts in its own right because its sequelae include pelvic inflammatory disease resulting in tubal infertility and ectopic pregnancy, fetal prematurity, and ophthalmia neonatorum.[3] The target reduction of 20% in the national rate of gonorrhoea from the 1990 level was reached by 1992.[4] This has been welcomed as an achievement of the NHS,[5,6] though recent statistics show a 5% increase in the total number of episodes of gonococcal infection treated at departments of genitourinary medicine in England between 1994 and 1995.[7]

The focus on trends in national rates of infection for the whole adult population masks known variations in the incidence of gonorrhoea by geography, age, and sex,[4] and it has been suggested that local targets defined by health authorities would be more useful.[8] Departments of genitourinary medicine, however, are open access clinics treating patients regardless of where they live. Hence the incidence of sexually transmitted infections in the population of a health authority cannot be calculated directly from the number of episodes recorded by local clinics. This protects individual patient confidentiality but limits the availability of basic epidemiological data about the scale of the problem, those who are at most risk, and where interventions are required.

In the United States, where some sexually transmitted infections are statutorily notifiable[9] and data are reported by age, sex, and ethnic group, there is at least a 10-fold excess of cases of gonorrhoea in African Americans compared with white, Hispanic, and all other ethnic groups. In urban areas the disparity is even greater.[10,11] Though socioeconomic deprivation is known to favour the spread of sexually transmitted infections[12] and disproportionate numbers of African Americans live in poverty,[13] the inequality in gonorrhoea rates persists after adjustment for socioeconomic confounding.[10]

In the inner London boroughs of Lambeth, Southwark, and Lewisham high numbers of gonococcal infections have been reported in heterosexual men of black African and black Caribbean ethnic groups.[14,15] We studied the epidemiology of gonorrhoea in Lambeth, Southwark, and Lewisham by using population based rates in order to aid the setting of appropriate local targets for interventions aimed at improving sexual health.

Methods

Ascertainment of cases

Sixteen departments of genitourinary medicine in south and central London were contacted (fig 1). A deadline for data collection was set three months after the end of the study period. By 31 March 1996 permission had been obtained and data collected from eight out of 10 clinics in south London and three out of six in central London.

Episodes of uncomplicated and complicated gonococcal infection diagnosed from 1 January 1994 to 31 December 1995 were identified from computerised or manual records by the diagnostic coding system used

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to report cases of sexually transmitted infections to the Department of Health (form KC60). KC60 codes B1, B1.1-3, B1.4a-c, and B5 were included, taking into account a change in the diagnostic categories introduced on 1 January 1995. Repeat episodes were defined as a new diagnosis made four weeks or more after a previous diagnosis in records with the same sex, date of birth, and postcode. When repeat episodes were detected only the first diagnosis in each 12 month calendar period was included in the analysis.

First episodes in adults aged 15-59 years were included in the final dataset only if the postcode (checked against a printed list provided by Lambeth, Southwark, and Lewisham Health Authority) was within the administrative boundaries of the boroughs. Records with no address were excluded. Incomplete postcodes were completed when possible from available information by using the Post Office directory.[16] Postcode, date of episode, sex, date of birth, ethnic group, country of birth or nationality, and whether episodes in men were acquired homosexually were entered as individual records in spreadsheet files protected by a password. To anonymise the records and ensure that deductive disclosure of individual identity was not possible date of birth and postcode were deleted from each record immediately after the exclusion of duplicate and repeat episodes and computerised allocation to an electoral ward.

Categorisation of cases by ethnic group into 1991 census categories[17] was attempted, but differences between clinics in classification systems recording race, ethnic group, nationality, or country of birth meant that only three broad groupings could be used. "White" included people recorded as white or Caucasian. Fourteen people with European, North American, or Australian country of birth were assumed to be white; "black" comprised those recorded as black, black African, black Caribbean, or black "other" and included three people with African or Caribbean country of birth or nationality; "other" comprised people from all other ethnic groups and nationalities. "UK" nationality or country of birth and those with no information were coded as unknown ethnic group.

Fig 1 Map of departments of genitourinary medicine in Greater London showing location of clinics surveyed

Denominator populations

Local base statistics from the 1991 census were used (table L06 (Crown copyright)) to estimate the population at risk in the 73 wards of Lambeth, Southwark, and Lewisham. The ward population was stratified by sex, age (15-19, 20-24, 25-29, 30-59 years), and ethnic group (white, black (black African, black Caribbean, black "other"), other (all other ethnic groups)). Undercoverage of the population by the census in inner London was corrected by using published adjustment factors specific for age and sex (appendix).[18]

Statistical analysis

Yearly incidence rates of gonorrhoea specific for age, sex, and ethnic group in residents of Lambeth, Southwark, and Lewisham per 100 000 population were calculated with 95% confidence intervals. Poisson regression models[19] were used to examine the relation between gonorrhoea, ethnic group, age, and sex before and after adjustment for confounding by socioeconomic deprivation. Individual measures of socioeconomic status were not available, so a ward level measure of underprivilege used by Lambeth, Southwark, and Lewisham Health Authority for the allocation of deprivation payments to general practitioners was applied to each case resident in the ward. The Jarman score is a weighted average of eight census derived variables which correlates with self reported morbidity[20] and all cause mortality.[21] A three level category corresponding to the standard cut off points for deprivation payments was used as a proxy for socioeconomic underprivilege—namely, 0-29.99 (no deprivation), 30-39.99 (low deprivation), and 40 or more (medium or high deprivation).

Evidence for effect modification by sex was examined by using a likelihood ratio χ^2 test. Rate ratios with 95% confidence intervals for the effect of age and ethnic group on gonorrhoea rates in men and women were calculated before and after adjustment for each other and for socioeconomic deprivation.

All statistical analyses were conducted with STATA (version 4.0, Austin, Texas).

Results

From 1 January 1994 to 31 December 1995, 2256 episodes of gonorrhoea were enumerated from 11 departments of genitourinary medicine. Data from 76 episodes with south London postcodes that could not be completed were excluded, leaving 2180 episodes in 1932 adults aged 15-59 years who were resident in Lambeth, Southwark, and Lewisham at the time of diagnosis. Of the 248 repeat episodes, two thirds occurred in men and three quarters in people from a black ethnic group. Forty six occurred in people who also had a recorded episode in the previous calendar year. This analysis therefore included 1978 first episodes of gonorrhoea in two calendar years. Information about heterosexual or homosexual acquisition of infection was absent from 46% of male records, so all cases in men were considered together.

Table 1 shows the distribution of cases in women and men.

Tables 2 and 3 give the age, sex, and ethnic specific rates of gonorrhoea in Lambeth, Southwark, and Lewisham. Overall rates were 138.3 and 291.9 cases yearly per 100 000 population aged 15-59 years for women and men respectively. For women in white and black ethnic groups the highest rates of infection were seen in teenagers (table 2). The highest age specific rates in men were for black men aged 20-24 years and white men aged 25-29 years (table 3). Rates for men and women from other ethnic groups were based on small numbers and not considered in detail. Rates were higher in men than in women at all ages except 15-19 years. In all age groups gonococcal infection was less common in white men and women than in men and women from black minority ethnic groups.

Table 1 Characteristics of patients with one or more episodes of gonorrhoea in Lambeth, Southwark, and Lewisham, 1 January 1994 to 31 December 1995

Characteristic	No (%) of women (total=646)	No (%) of men (total=1332)
Age (years)		
15-19	213 (33)	142 (11)
20-24	222 (34)	331 (25)
25-29	125 (19)	420 (31)
30-59	86 (13)	439 (33)
Ethnic group		
White	126 (20)	304 (23)
Black	424 (66)	837 (63)
Other	32 (5)	35 (3)
Unknown	64 (10)	156 (12)
Deprivation level of ward of residence		
None	164 (25)	323 (24)
Low	238 (37)	509 (38)
Medium or high	244 (38)	500 (38)
Genitourinary department attended		
Lambeth, Southwark and Lewisham (three clinics)	578 (89)	1069 (80)
Outside Lambeth, Southwark, and Lewisham (eight clinics)	68 (11)	263 (20)

Age, ethnic grouping, and deprivation

As suggested by the stratified gonorrhoea rates there was strong statistical evidence for an interaction between sex and ethnic group ($P=0.0006$) and between sex and age ($P<0.0001$) (table 4). Regression analyses are therefore presented separately for women and men.

Women—Rates of gonorrhoea were strongly associated with age and ethnic group. Young age was the strongest risk factor, teenagers being 15 times more likely than women over 30 to experience gonorrhoea after adjustment for confounders. Adjusting for age and deprivation score had only a small effect on the effect of ethnic group. Women from black minority ethnic groups had around 10 times the rate of gonococcal infection seen in white women.

Men—As in women, men over the age of 30 had the lowest rate of gonorrhoea but the adjusted rate ratios for younger men did not show a strong gradient, ranging from 2.0 to 2.6. The effect of ethnic group in men was similar to that for women. After adjustment for age and Jarman score men from black minority ethnic groups were 11 times more likely than white men to experience gonorrhoea.

For both men and women the effect of living in a ward with any deprivation compared with wards with no deprivation (defined by Jarman score) was modest and unaffected by further adjustment for age and ethnic group.

Discussion

Methodological considerations

These analyses underestimate the frequency of gonorrhoea in the study population. Firstly, patients with no address and those attending non-participating clinics could not be included. Secondly, only genitourinary clinics complete form KC60. Microbiology records for 1994-5 from a local hospital with no genitourinary department identified only 45 episodes of gonorrhoea from all other clinical settings (G Rao, personal communication). Thirdly, repeat episodes were excluded from multivariate analyses because these observations are not independent of one another. For consistency they were also excluded from descriptive

Table 2 Incidence rates (95% confidence intervals) of gonorrhoea in female residents of Lambeth, Southwark, and Lewisham per 100 000 population yearly by age and ethnic group, 1994-5

	White ethnic group		Black ethnic group		Other ethnic groups	
Age (years)	No of cases	Rate	No of cases	Rate	No of cases	Rate
15-19	41	171.4 (111.2 to 264.2)	149	1710.7 (1363.2 to 2146.8)	8	198.0 (74.3 to 527.6)
20-24	48	90.7 (60.8 to 135.3)	142	888.6 (704.2 to 1121.3)	12	232.5 (104.4 to 517.5)
25-29	21	30.3 (16.5 to 55.5)	77	321.9 (234.7 to 441.4)	8	1040.4 (52.7 to 374.0)
30-59	16	8.4 (4.2 to 16.8)	56	116.6 (80.5 to 168.9)	4	23.2 (5.8 to 93.0)
All ages	126	37.4 (31.4 to 44.6)	424	438.8 (383.5 to 502.0)	32	99.7 (70.5 to 140.9)

Table 3 Incidence rates (95% confidence intervals) of gonorrhoea in male residents of Lambeth, Southwark, and Lewisham per 100 000 men yearly by age and ethnic group, 1994-5

	White ethnic group		Black ethnic group		Other ethnic groups	
Age (years)	No of cases	Rate	No of cases	Rate	No of cases	Rate
15-19	19	80.3 (42.5 to 151.8)	113	1342.7 (1034.5 to 1742.7)	5	122.2 (35.4 to 422.1)
20-24	62	121.2 (85.3 to 172.4)	232	1685.6 (1405.1 to 2022.0)	9	166.7 (66.2 to 419.9)
25-29	106	142.2 (108.6 to 186.1)	253	1330.9 (1118.0 to 1584.2)	10	172.8 (71.9 to 415.1)
30-59	117	60.5 (46.8 to 78.1)	239	598.6 (500.3 to 716.1)	11	62.6 (27.1 to 144.3)
All ages	304	88.7 (79.2 to 99.2)	837	1031.8 (964.2 to 1104.2)	35	106.5 (76.5 to 148.4)

Table 4 Rate ratios (95% confidence intervals) for effects of age, ethnic group, and deprivation score on rates of gonorrhoea in women and men in Lambeth, Southwark, and Lewisham

	Women				Men			
Variable	**Unadjusted**	**P**	**Adjusted†**	**P**	**Unadjusted**	**P**	**Adjusted†**	**P**
Age (years)								
30-59	1		1		1		1	
25-29	3.5 (2.6 to 4.7)	<0.0001	3.0 (2.3 to 4.1)	<0.0001	2.5 (2.2 to 2.9)	<0.0001	2.3 (2.0 to 2.6)	<0.0001
20-24	9.0 (6.9 to 11.7)	<0.0001	8.3 (6.4 to 10.8)	<0.0001	3.0 (2.5 to 3.4)	<0.0001	2.6 (2.2 to 3.0)	<0.0001
15-19	17.8 (13.6 to 23.1)	<0.0001	15.2 (11.6 to 19.7)	<0.0001	2.6 (2.1 to 3.2)	<0.0001	2.0 (1.7 to 2.5)	<0.0001
Ethnic group								
White	1		1		1		1	
Black	11.7 (9.6 to 14.3)	<0.0001	10.5 (8.6 to 12.8)	<0.0001	11.6 (10.2 to 13.3)	<0.0001	11.0 (9.47 to 12.6)	<0.0001
Other	3.2 (2.2 to 4.8)	<0.0001	2.3 (1.5 to 3.4)	<0.0001	1.6 (1.3 to 1.8)	0.304	1.2 (0.8 to 1.7)	0.354
Deprivation score								
None	1		1		1		1	
Low	1.5 (1.2 to 1.8)	<0.0001	1.5 (1.2 to 1.9)	<0.0001	1.6 (1.4 to 1.9)	<0.0001	1.6 (1.4 to 1.9)	<0.0001
Medium or high	1.5 (1.2 to 1.8)	<0.0001	1.5 (1.2 to 1.9)	<0.0001	1.6 (1.3 to 1.8)	<0.0001	1.6 (1.4 to 1.9)	<0.0001

†Adjusted for ethnic group, Jarman score, and age.

analyses, and crude rates represent the number of people affected per 100 000 yearly. Repeat episodes occurred more often in people from black ethnic groups,[14 15] so exclusion may reduce the magnitude of effect of ethnic group but does not alter the conclusions.

The Jarman score was a convenient proxy for underprivilege at ward level. The proportion of households headed by a person born in the new Commonwealth or Pakistan is one component of the score, even if with the lowest weighting.[21] Thus the ecological association between gonorrhoea and the Jarman score could simply reflect the ethnic composition of ward populations. Analyses of the relation between gonorrhoea rates and single indicators of socioeconomic status and other validated deprivation scores[21] are now planned.

The hidden epidemic

Geography—In 1993 national reported rates of 22 and 43 gonorrhoea cases per 100 000 women and men aged 15-59 years respectively were below the *Health of the Nation* target.[4] Among residents of Lambeth, Southwark, and Lewisham in 1994-5 the corresponding rates were 138 and 292 cases per 100 000 yearly. Roughly 11% of all episodes of gonorrhoea reported from departments of genitourinary medicine in England in 1994-5[7] occurred in residents of Lambeth, Southwark, and Lewisham, who accounted for 1.4% of the population aged 15-59 years (1991 census data). High rates have also been recorded in the West Midlands[22] and Leeds (C J N Lacey, D Bensley, D Merrick, I Fairley, paper presented at the 11th meeting of the International Society for Sexually Transmitted Diseases Research, New Orleans, August 1995). Presentation of national rates of gonorrhoea thus hides the disproportionate contributions of a few geographic foci. These findings should not be extrapolated to other, particularly viral sexually transmitted infections, which are more uniformly distributed.[23]

Sex—Twice as many cases of gonorrhoea in men as in women were identified. Including infections acquired through sex between men does not account for this disparity. About a quarter of episodes for which information was available were acquired homosexually. When this proportion of all male cases is subtracted the male to female ratio is still 1.5:1, indicating that women are remaining untreated in the community. The highest rates of diagnosed gonorrhoea—that is, in teenage women from black ethnic groups—would be two to three times higher if the denominator was restricted to women who were sexually experienced.[24] These young women are also at risk of pelvic inflammatory disease, tubal infertility, and ectopic pregnancy.

Ethnic grouping—Information about ethnic grouping was too inconsistent to allow distinctions to be made within broad groups. The results should not therefore be applied to individual minority populations but they highlight inequalities in sexual health which require investigation. Increased monitoring of ethnicity in health service settings using census categories[17] should improve the quality of these data. Knowledge of cultural, social, and economic influences on sexual health is scarce in the United Kingdom. Pejorative studies of gonorrhoea in immigrants from the Common-

Key messages

- Presentation of national rates of gonorrhoea hide the disproportionate contribution of small geographic foci with exceptionally high incidence rates
- Gonorrhoea occurs largely in teenage women and young adult men; calculated rates in adolescents of both sexes would be even higher if the denominator was restricted to those who are sexually active
- Large inequalities in the incidence of gonorrhoea exist between ethnic groups, and these persist after adjusting for socioeconomic underprivilege
- The complex reasons for ethnic inequalities in sexual health need to be elucidated so that effective interventions can be designed and implemented
- The resource implications of underdiagnosis of gonorrhoea in young women for the management of long term sequelae, including ectopic pregnancies and tubal infertility, should be examined

wealth in the 1950s and 1960s[25-27] have probably inhibited useful research in more recent years.

Recommendations

In the United States, Healthy People 2000—the equivalent of Health of the Nation—has set ethnic specific targets[9] and directed resources towards reducing the disparity in rates of sexually transmitted infections. As a start in the United Kingdom accurate information should be disseminated to the community, health service providers, purchasers, and policy makers. The detection of inequalities in rates of gonorrhoea experienced by members of minority ethnic groups should be used positively to argue for resources to investigate the reasons for these disparities and to intervene appropriately and effectively.

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Appendix

Adjustment factors for estimated census undercoverage in inner London by age and sex[18]

Age (years)	Women	Men
15-19	1.01	1.03
20-24	1.05	1.19
25-29	1.06	1.22
30-34	1.03	1.11
35-39	1.00	1.02
40-44	1.00	1.02
45-59	1.00	1.00

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My most unfortunate mistake

A memorable viva

Third year at medical school at Edinburgh university in the 1980s was not a stressful year. We had just passed the notoriously difficult second MB and were floating on a wave of success. Several of my contemporaries did not attend classes at all in the first term. I usually managed to attend once or twice a week. This sabbatical period was put to good use exploring the hills and glens of Scotland and generally "finding oneself." Much sociology and other odd subjects (most unrelated to medicine) were learnt during this period by the oral tradition.

Unfortunately at the end of the second term an exam was approaching. I found myself sitting at the other side of a desk from one of our senior tutors. I had just invented my latest theory on the classification of bacteria. He was not looking impressed.

"Tell me," he said. "Which book have you been using for revision?"

He picked up a book from his desk and casually flicked its pages. I noticed that this was one of the books on our reading list. Six months previously I had chosen to buy its competitor in preference. The book I had bought covered the same topics but in greater detail than the book held by my tutor. Unfortunately I was still suffering from the delusion that buying a book in some way imparts knowledge without actually reading the book.

Tact was not one of my stronger personality traits.

"I don't like that book," I said. "It's just a book of lists."

As I tried to retrieve my last sentence I noted that the name of the author and of my tutor were one and the same.

After that, as you could expect, the interview deteriorated. Several difficult questions and about five minutes later the tutor made his assessment.

"I don't think your knowledge quite merits a pass, perhaps what you need is a book of lists."

Colin McCartney, *anaesthetic registrar, Aberdeen*

Poverty or income inequality as predictor of mortality: longitudinal cohort study

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Abstract

Objective: To determine the effect of inequality in income between communities independent of household income on individual all cause mortality in the United States.

Design: Longitudinal cohort study.

Subjects: A nationally representative sample of 14 407 people aged 25-74 years in the United States from the first national health and nutrition examination survey.

Setting: Subjects were followed from initial interview in 1971-5 until 1987. Complete follow up information was available for 92.2% of the sample.

Main outcome measures: Relation between both household income and income inequality in community of residence and individual all cause mortality at follow up was examined with Cox proportional hazards survival analysis.

Results: Community income inequality showed a significant association with subsequent community mortality, and with individual mortality after adjustment for age, sex, and mean income in the community of residence. After adjustment for individual household income, however, the association with mortality was lost.

Conclusions: In this nationally representative American sample, family income, but not community income inequality, independently predicts mortality. Previously reported ecological associations between income inequality and mortality may reflect confounding between individual family income and mortality.

Introduction

Studies have documented the powerful association between a person's socioeconomic status and mortality.[1-4] Recently, ecological studies have suggested that income inequality is also correlated with overall mortality.[5,6] Wilkinson reported a correlation of −0.81 between national income inequality among 11 industrialised countries and national life expectancy after controlling for gross national product per head.[7] Comparable findings were recently reported using data at state level from the United States. Kaplan et al noted that state income inequality adjusted for state median income was significantly correlated ($r=0.62$) with all cause mortality, age specific mortality, low birth rate, homicide, violent crime, work disability, expenditures on medical care, and police protection.[8] Kennedy et al reported that state income inequality adjusted for levels of poverty was strongly correlated ($r=0.54$) with age adjusted total mortality, infant mortality, coronary heart disease, malignant neoplasms, and homicide.[9] Ben-Shlomo et al showed significant effects for income inequality measured at the British ward level on area mortality.[10]

These ecological or population level studies suggest that the relation between income and mortality in developed countries is a relative phenomenon. In other words, income inequality between countries, states, or communities, is more strongly associated with health than is poverty or mean per capita income. Income equality may be associated with mortality in several ways. Firstly, income inequality may affect health via cognitive processes such as perceived deprivation that promote hopelessness, hostility, or risk taking behaviour.[11-16] Secondly, income inequality may be a measure or cause, or both, of social forces such as reduced social cohesion that affect health.[17] Thirdly, income inequality may be a marker of a government's underinvestment in human resources.[18] Lastly, the association between income inequality and mortality may simply represent confounding by family income at the individual level.

Studies reporting an association between income inequality and mortality have provided limited insight into the nature of this relation. Conclusions from previous studies are limited by potential "ecological fallacy" because they cannot adequately control for confounding at the individual level.[19] In other words, the observed relation between income inequality and mortality observed at a population level may simply represent inadequately measured rates of income differences at the individual level. No published studies have specifically examined the effect of income inequality on mortality after adjustment for income at the individual level.

We examined whether inequality in income between communities predicts future individual mortality independent of family income. We used data from a nationally representative prospective cohort from the United States to assess whether residence in communities with greater income inequality was independently associated with mortality at follow up.

Methods

Source of data

The first national health and nutrition examination survey (NHANES I), conducted between 1971 and 1975 in the United States, collected sociodemographic data from multiple national probability samples of the civilian non-institutionalised population of adults aged 25 to 74 years.[20,21] The epidemiological follow up study (NHEFS) collected mortality data through follow up surveys conducted in 1982, 1984, 1986, and 1987.[22,23] Follow up data were derived from interview surveys, medical records from healthcare institutions, and all death certificates. The age, race, and sex specific mortality of the follow up cohort is comparable to that experienced by the American population.[23] In all, 14 407 people were in the original survey, 3.4% of whom had missing information on family income; mortality status at follow up was ascertained on 95.8%

of the people for whom such information was available.

The first survey used multistage stratified probability samples of people from 105 areas or primary sampling units in the United States. The areas approximated to counties or combined county areas. People living in areas of poverty, women of childbearing age, and elderly people were oversampled (surveyed at disproportionately higher rates). A mean of 131 (range 48-323) people were surveyed in each primary sampling unit. The revised weights provided on the 1987 follow up study's "public use" tapes were used to adjust for survey oversampling and non-response to yield population estimates for each community surveyed.

Measure of family income

Household income was assessed through response to a single question in the first national health and nutrition examination survey. The question asked subjects to say which of 12 income groups represented their total family income for the previous 12 months, including all sources of income, such as wages, salaries, social security or retirement benefits, help from relatives, and rent from property. The income categories ranged from under $1000 to $25 000 and over. Subjects were assigned to the mean value within their income category. Subjects in the highest income category (4.6%) were assigned a mean value by extrapolation.

Measure of income inequality

Several indices of community income inequality have been described, though the relation of each index to mortality risk is similar.[9] We used an index that estimates the proportion of total income earned by the poorer half of the population in the area. The denominator for this index is the total aggregate income in the community (primary sampling unit), and the numerator is the aggregate income in the community earned by the poorer half of the population. The income inequality within the communities ranged from 0.18 to 0.37.

Statistical analysis

Multilevel modelling is required to avoid the problems of clustering within a group.[24] We used the statistical package SUDAAN, which uses a Taylor series approximation method to compute variances that allow adjustment for multistage probability sampling.[25] A Cox proportional hazard survival analysis was performed that included the index of community income inequality, household income, family size, sex, and age as covariates in the predictive model for mortality. The assumptions of the model were tested and found valid.

Results

Older age, residence at the time of the interview in a community with greater income inequality, and lower mean community income were all associated with the proportion of people in the community dying during follow up (table 1). Survival analysis showed that survival adjusted for age, sex, and family size was associated with income inequality (hazard ratio = 0.23, 95% confidence interval 0.06 to 0.86); additional adjustment for mean community income did not greatly affect this relation (0.31, 0.10 to 0.90). However, after adjustment for household income, no significant relation between income inequality and mortality was evident (table 2). An analysis that excluded the income inequality measure showed no change in the effect size of income on mortality (0.97, 0.96 to 0.98).

Table 1 Bivariate correlations between proportion of people in community dying during follow up and community level sociodemographic characteristics (n=105)

Variable (by community)	Mean (SD)	Correlation	P value
Age	46.61 (2.36)	0.64	0.001
Male (proportion)	0.47 (0.07)	0.13	0.184
Income ($000s)	12.03 (2.62)	–0.48	0.001
Community income inequality*	0.28 (0.03)	–0.34	0.004
Death (proportion)	0.15 (0.06)	NA	

NA=not applicable.
*Proportion of total area income earned by the poorer half of area residents.

Table 2 Survival analysis of factors affecting mortality hazard during follow up (n=13 280)

Risk factor	Hazard ratio (95% CI)	P value
Age	1.09 (1.08 to 1.09)	<0.001
Female sex	0.53 (0.49 to 0.57)	<0.001
Household income ($000s)	0.97 (0.96 to 0.98)	<0.001
Community income inequality*	0.81 (0.22 to 2.92)	0.752

Analysis is also adjusted for family size. Hazard ratios indicate the increase in risk of mortality during follow up based on a unit change in the risk factor.
*Proportion of total area income earned by the poorer half of community residents.

Discussion

Analyses of data from a nationally representative prospective American cohort study show that individually measured family income strongly confounds the relation between community income inequality and mortality. Although aggregated data at the individual level simulated the findings of previous ecological studies,[7-10] community income inequality did not independently predict mortality after adjustment for family income. Conversely, exclusion of the variable of community income inequality did not affect the relation between family income and subsequent mortality. These findings suggest that the effect of income inequality reported in ecological studies may result from confounding by income at the individual level.

Our findings imply that income, as a measure of access to resources, and not relative inequality, better explains the relation between income and mortality. Psychological or social factors related to income inequality may nevertheless have important health effects, as Wilkinson suggests.[17] We believe, however, that existing studies have not adequately tested this hypothesis. Future studies of the inequality hypothesis should control for income at the individual level and use direct measures of factors such as social cohesion and perceived socioeconomic deprivation to advance our understanding of the relative contribution of these factors to health.

These findings are subject to several important caveats. The appropriate unit of analysis for measuring income inequality is not known. Ecological studies have shown effects of inequality at the national level,[7]

American state level,[8 9] and British ward level.[10] We used community (mostly American counties) income inequality as our unit of analysis and showed significant effects for income inequality in a simulated ecological analysis. The validity of our analysis is supported by the significant correlation between community income inequality and community mortality rates after adjustment for mean community income. Although the magnitude of this correlation was smaller than that reported in previous ecological studies, this may represent less non-differential misclassification bias.[26] Because our ecological analysis used prospective data that had been carefully collected at the individual level, there may have been less misclassification than in ecological studies that have used national or state cross sectional data. Brenner et al showed that non-differential exposure misclassification in ecological studies as opposed to individual level studies can lead to significant overestimation of effects.[26] Further support for the validity of our findings is provided by preliminary data from the panel on income dynamics from the United States. In this study state income inequality showed no effect on mortality after individually measured income was controlled for (G Duncan, personal communication).

Limitations

Still larger units of analysis might yield different results. This hypothesis is particularly plausible if it is assumed that income inequality is simply a proxy for national policies that promote general social welfare and health.[8 27] However, if income inequality is assumed to influence health directly via cognitive processes then the appropriate unit remains speculative. For example, if people judge their socioeconomic status relative to that of their neighbours, then the community may be the appropriate level of analysis, but if they use national media sources as a frame of reference then analyses should focus on the national level. A stronger conceptual framework is needed to guide future studies in this area and also to provide direct testing of the hypothesis that individual perceptions of relative socioeconomic standing influence health.

These findings are limited by the area sampling methodology used by the first national health and nutrition examination survey. Although the survey was designed as a representative sample of the American population, sampling within communities was not random. The cluster sampling strategy underestimates the true variability in each community, thus underestimating community income inequality. Despite this bias, we found significant effects for income inequality in the ecological but not individual level analysis.

Our analysis does not account for an individual's relocation from one community to another during the study. Nor does our analysis account for increasing levels of income inequality in the United States during the study.[28] Such misclassification bias tends to overestimate the effects observed in an ecological study and to underestimate the effects observed in an individual level analysis. This bias operates similarly for family income. Both family size and income may change considerably over time, resulting in further misclassification bias. In addition, a selective bias resulting from greater loss to follow up among poorer people tends to understate the effect of poverty on mortality. Although each of these biases underestimates the effect of family income on mortality, we none the less found significant effects for family income.

Key messages

- Ecological studies have documented a powerful relation between national, state, or community indices of inequality and mortality at the population level
- These studies have not adequately controlled for confounding by family income measured at the individual level
- This study, using data from a nationally representative sample from the United States, showed no relation between community income inequality and mortality after adjustment for family income
- Poverty, not community income inequality, determines subsequent mortality; further study is needed to determine whether these findings apply to national or state-wide income inequality

Colinearity between family income and community income inequality may have masked the independent effect of community income inequality on mortality. The finding that the relation between family income and mortality was unaffected by adjustment for community income inequality suggests, at least, that family income is a far more powerful predictor of health status than community income inequality. Although our analysis does not exclude a modest effect of community income inequality on health, these findings militate against a large effect.

Conclusion

These findings suggest that community income inequality does not have large effects on mortality independent of the effects of family income. However, income inequality and family income are closely intertwined. Countries, states, or communities with large income inequalities are likely to have more poverty. Countries whose explicit goal has been eradication of poverty also have less income inequality. Thus, whether public policy focuses primarily on the elimination of poverty or on reduction in income disparity, neither goal is likely to be achieved in the absence of the other.

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Commentary: Income inequality summarises the health burden of individual relative deprivation

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Fiscella and Franks claim that the relation between societal mortality and income inequality is an ecological fallacy, but in fact their data makes a useful contribution wholly consistent with that relation. Their data shows that individual income levels largely account for the income inequality relation in American counties. But the relation between individual income and mortality is primarily an effect of *relative* income.[1 2] As inequality defines relative income, what the paper suggests is that the relation between population mortality and inequality reflects the health implications of a person's relative income rather than any effects of wider societal processes.

The nub of the problem is the assumption that mortality is affected by absolute levels of income—as if $20 000 bought a given amount of health regardless of the incomes of the rest of society. But in practice how much health it buys depends on whether it makes you rich or poor compared with the rest of society. The reason that the affluence of the United States does not lead to mortality rates as low as in many poorer countries is not because the United States has more people in *absolute* poverty. Indeed, at the state level the association between mortality and inequality remains after absolute poverty is controlled for.[3] The problem is that the United States has more relative poverty, and this is likely to affect health, mainly—as this paper shows—through individual relative income.

The major health inequalities that accompany socioeconomic differences across the social hierarchy are now widely recognised. What the income distribution relation tells us is that health is worse when there is greater inequality across this social gradient. Rather than hinging on some other dimension of inequality operating between neighbours or within the local community, the effect of income inequality is almost certainly tied up with the central sociological processes of social stratification.

That absolute income levels are no longer important in the developed world is shown by the lack of a strong association between mortality and average incomes among developed nations or American states.[2-5] The weak correlation with median state income disappears completely after income distribution within states is controlled for.[1] In contrast, *within* countries or states income is closely related to mortality because it measures differences in socioeconomic status.[2]

In smaller areas of analysis, however, the situation is reversed: between small areas median income *is* related to mortality, whereas income distribution within them is not.[6-8] That is because the salient inequality lies in the societal system of social stratification.[5] In the smallest areas the social heterogeneity, which makes inequality important, is lost. At the extreme, in small, socially homogeneous, one class neighbourhoods, the inequalities that matter would all be between, not within, neighbourhoods. Differences in their median community income would do all the work in relation to mortality, while inequality within them would do none. Individual income matters because it is an indicator of position in relation to the societal system of social stratification. After all, Harlem's appalling health does not result from

the inequalities within Harlem but from its deprivation in relation to the United States outside Harlem.[9]

At the intermediate level of American counties used by Fiscella and Franks, some of the social heterogeneity that accounts for the explanatory power of inequality lies within them and some between them; so income inequality explains less variation in mortality here than it does at the state or national level, but more than it would in smaller areas. Thus what their analysis really shows is that the effect of the inequality attributable to the residual social heterogeneity within counties can be explained by the effects of peoples' incomes relative to the wider society.

This kind of analysis cannot finally tell us whether there are also wider knock-on effects to the rest of society from increased relative deprivation—perhaps arising from the higher levels of homicide, accidents, and alcohol related deaths associated with less egalitarian societies.[9-11] That would be possible only if the relation between individual income and health could be measured independently of its determination in a particular society with its particular level of income inequality.

What is important, however, is that national mortality rates can be lowered by redistributing income. Average health is improved not by simply redistributing a given amount of health (by redistributing the current stock of health producing goods), but by reducing the psychosocial burden of relative deprivation. Fiscella and Franks's interpretation of their results implies a steeply curved relation between *absolute* income and mortality. If such a relation existed then economic growth without redistribution would rapidly narrow health inequalities. As animal models have shown, subordinate social status has health consequences even when the physical environment and diet are invariant.[12 13]

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BMJ audit: time to decision and publication

We aim to make a decision on publication within eight weeks (56 days); to reject papers that are unsuitable for external peer review within two weeks (14 days); and to publish a paper within eight weeks of acceptance.

Between July and December last year we made a decision within 56 days for 76% of all papers submitted (1491/1951) and for 53% of those accepted (92/173). We accepted 56% within 66 days, and the mean time to accept a paper was 88 days. We met our target of rejecting papers without peer review within 14 days for 32% of papers (363/1134); 62% were rejected within 24 days.

Overall we published 29% of papers within eight weeks of acceptance, 52% within 10 weeks, and 66% within 12 weeks. Of research papers, however, we published 39% within eight weeks, 73% within 10 weeks, and 88% within 12 weeks.

Table 1 Results of *BMJ* audits. Values are percentages unless stated otherwise

	Decision within 56 days		Accepted papers		Rejected papers (no peer review)		Publication after acceptance within:		
Audit	All papers	Accepted papers	Decision within 66 days	Mean time to decision (days)	Decision within 24 days	Mean time to decision (days)	8 weeks	10 weeks	12 weeks
1993:									
Jan-June	88	73	85	41	76	19	38	75	95
July-Dec	86	62	75	50	84	18	27	66	85
1994:									
Jan-June	88	64	76	48	84	18	13	24	57
July-Dec	83	64	73	51	73	21	40	67	87
1995:									
Jan-June	72	41	53	69	56	26	38	60	76
July-Dec	73	34	43	81	65	22	32	50	73
1996:									
Jan-June	81	43	59	59	65	24	19	35	53
July-Dec	76	53	56	88	62	23	29	52	66

Does it matter who requests necropsies? Prospective study of effect of clinical audit on rate of requests

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Despite the potential benefits of postmortem examination, rates are declining throughout the world.[1] Reasons for this decline include the reluctance among doctors to request,[2] relatives to give permission for,[3] and pathologists to perform necropsies. This trend might be reversed by using medical education to change doctors' attitudes; delegating the task of requesting necropsies to other staff[4]; and increasing public awareness as to the potential benefits of necropsies. We prospectively studied the effect of clinical audit on necropsy rate and the subsequent effect of a patient affairs officer assuming responsibility for requesting necropsies.

Methods and results

Since March 1991 we have audited the necropsy rate in a 112 bed department of acute medicine for the elderly. The results of an initial six month audit and the potential benefits of and possible barriers to achieving a high necropsy rate were discussed with members of the department, and the departmental policy of obtaining the highest possible necropsy rate was reinforced. The impact of our audit programme on the rate of necropsies obtained by medical staff was then observed over six months, during which we recorded request and refusal rates. On completion of this audit cycle, the patient affairs officer (JRJ) accepted responsibility for requesting necropsies as she believed that she could achieve a higher necropsy rate than the medical staff. Subsequent audit compared her performance with that previously achieved by medical staff. Over three successive years we audited the annual performance of the patient affairs officer and looked at the effect of relatives' refusal to give permission for necropsy.

Clinical audit had a marginal effect on the rate of necropsies obtained by doctors, which increased from 22.3% to 28.0%. There was a more substantial increase to 46.2% when the patient affairs officer requested necropsies (table 1), due mainly to an increased rate of requests (refusal rates for medical staff and the patient affairs officer are comparable (38.8% *v* 36.8%)). The rate of requests by the patient affairs officer showed a consistent annual increase, rising from 64.3% to 79.6%, but the necropsy rate plateaued at around 51%, apparently because of a stable refusal rate of about one third.

Comment

The marginal effects of audit on the necropsy rate obtained by junior doctors suggest that they are reluctant to request necropsies. Many hospitals in the United States, but few in Britain, delegate the task of requesting necropsies to non-medical staff; we are not aware of reports of their performance or of comparisons with medical staff. Our patient affairs officer's approach to requesting necropsies has proved satisfactory to the recently bereaved relatives and has gained the confidence and support of the medical staff. It is likely that she is more effective than medical staff in requesting necropsies because of her enthusiasm; there is no conflict of interest as she has not had contact with the deceased patients or their relatives; she may have better communication skills; and there are fewer demands on her time.

Our study also shows that relatives' refusal to give permission for necropsy is an important limiting factor in achieving a high necropsy rate. This is independent of the process of requesting the examination.

A minimum necropsy rate of 35% has been recommended for clinical audit.[5] To achieve this, necropsies must be requested on more than half of deaths as about a third of requests are likely to be refused. Our results show that a high necropsy request rate is more likely to be achieved by non-medical staff. A further increase in this rate is unlikely unless public attitudes to necropsy are changed.

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Table 1 Effects of clinical audit and requests by patient affairs officer on rates of request, refusal, and performance of necropsies

Review period	Total No of deaths	No (%) of coroner's necropsies	No (%) of necropsies requested	No (%) of refusals	No (%) of necropsies performed*
Six month review (1 May-31 Oct):					
1991 - doctors before audit	184	10 (5.4)			41 (22.3)
1992 - doctors after audit	255	10 (3.9)	116 (45.5)	45 (38.8)	71 (28.0)
1993 - patient affairs officer	223	9 (4.2)	162 (72.6)	59 (36.8)	103 (46.2)
Annual review (1 Nov-31 Oct):					
1992-3 - patient affairs officer	551	23 (4.2)	353 (64.3)	123 (35.8)	230 (41.4)
1993-4 - patient affairs officer	543	22 (4.1)	400 (73.7)	119 (29.8)	281 (51.8)
1994-5 - patient affairs officer	520	26 (5.0)	414 (79.6)	150 (36.4)	264 (50.8)

*Excluding coroner's necropsies.